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Increased mortality in COPD among construction workers exposed to inorganic dust

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ABSTRACT: The aim of this study was to find out if occupational exposure to dust, fumes or gases, especially among never-smokers, increased the mortality from chronic obstructive pulmonary disease (COPD).

A cohort of 317,629 Swedish male construction workers was followed from 1971 to 1999. Exposure to inorganic dust (asbestos, man-made mineral fibres, dust from cement, concrete and quartz), gases and irritants (epoxy resins, isocyanates and organic solvents), fumes (asphalt fumes, diesel exhaust and metal fumes), and wood dust was based on a job-exposure matrix. An internal control group with "unexposed" construction workers was used, and the analyses were adjusted for age and smoking.

When all subjects were analysed, there was an increased mortality from COPD among those with any airborne exposure (relative risk 1.12 (95% confidence interval (CI) 1.03–1.22)). In a Poisson regression model, including smoking, age and the major exposure groups, exposure to inorganic dust was associated with an increased risk (hazard ratio (HR) 1.10 (95% CI 1.06–1.14)), especially among never-smokers (HR 2.30 (95% CI 1.07–4.96)). The fraction of COPD among the exposed attributable to any airborne exposure was estimated as 10.7% overall and 52.6% among never-smokers.

In conclusion, occupational exposure among construction workers increases mortality due to chronic obstructive pulmonary disease, even among never-smokers.
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While there is consensus that cigarette smoking is a specific cause of chronic obstructive pulmonary disease (COPD), the association between occupational exposures and COPD has not been as straightforward. However, in recent years, an increasing consensus about the risk associated with dusty work has emerged [1–4]. One problem has been that measuring COPD in epidemiological studies is not simple, as no consensus exists regarding the epidemiological definition of COPD.

Annual decline of lung function has been used as one proxy variable for COPD. In longitudinal studies of occupationally exposed cohorts, the annual decline in forced expiratory volume in one second has been found to be related to gas, dust and fume exposure [5–10]. The reported declines were ~7–8 $mL \cdot yr^{-1}$, after adjustment for age and smoking.

Studies of coal miners have shown an increased mortality due to bronchitis and emphysema [11–13], especially centrilobular emphysema [14–16]. A relationship between dust exposure and degree of emphysema has been found in studies of coal and hard-rock miners [17, 18]. Gold miners seem to have a greater risk of dying from COPD than coal miners, which may be due to the higher silica contents in gold mine dust [2].

Regarding other kinds of occupational dust exposure, the literature is scanty. ROBINSON *et al.* [19] analysed the mortality among 27,362 US carpenters' union members. The study was not controlled for smoking habits and they observed an increased mortality due to emphysema among construction carpenters. SJÖGREN *et al.* [20], in a register-based study, reported that agricultural workers had increased mortality due to COPD. Hence, there is a need for powerful longitudinal studies evaluating the risk of COPD in groups occupationally exposed to other kinds of dust, not only from gold mines and coal mines. Here, the authors report the results of a prospective cohort study of 317,629 male construction workers exposed to different types of dust. The more specific aim of the study was to elucidate whether occupational exposure to inorganic dust, smoke or fume increased the risk for death from COPD, taking smoking habits into account.

Methods

In 1968, the Swedish Foundation for Occupational Safety and Health (Bygghälsan) was established to coordinate all activities concerning occupational health among Swedish construction workers. Construction workers were invited to health examinations at intervals of 2–5 yrs. Although the programme was voluntary, at least 80% of eligible workers participated at least once. Data from the health examinations, including occupational titles and smoking habits, were registered in a central database established in the early 1970s, as previously described [21–23]. Through the personal identity number and a linkage with the National Cause of Death Register, it was possible to identify subjects who died (underlying cause) from COPD. The diagnoses from the International Classification of Diseases (ICD) were used, as

follows: from ICD 8, 490–492; from ICD 9, 490–492 and 494–496; and from ICD 10, J43–J44. Each worker was followed from entry into cohort (earliest 1 January 1971) until 31 December 1999. The loss of subjects was low (0.15%) and such persons were excluded from the analysis. Those examined before the age of 15 yrs or after the age of 67 yrs were also excluded.

The smoking habit at the time of the first health examination was used. When information on smoking was lacking, information from a later visit was used. Hence, smoking was classified as never-smokers, exsmokers, smokers and unknown smoking habits. The occupational title at the time of the first health examination was used. If the title included jobs that were often used for replacement of sick persons, *e.g.* storage worker or cleaner, the individual was excluded from further analysis. Most workers remained in the same occupation. For example, among the workers who participated in repeated examinations, 74% reported the same occupation. The exposed group consisted of 200,735 males. The reference group consisted of all males in jobs that were assessed as nonexposed in the job-exposure matrix (see below), 116,894 males in total. The study was approved by the committee of ethics at Umeå University (Umeå, Sweden).

Exposure assessment

A job-exposure matrix was developed for selected exposures as previously described [24]. In brief, the exposure assessment was based on a previous survey from the mid-1970s [25], where each occupation was studied at visits to approximately five different sites in different geographical regions of Sweden. More detailed information about this procedure can be obtained from the corresponding author. Each factor was graded on a 0–5 scale, by a group of occupational hygienists and physicians. Level 3 corresponded to the hygienic threshold value at the time of the study. When no such limits were applicable, level 3 corresponded to an exposure that at that time was considered "acceptable". Exposures to asbestos, asphalt fumes, cement dust, concrete dust, diesel exhaust, epoxy resins, isocyanates, man-made mineral fibres, metal fumes, organic solvents, quartz dust and wood dust were assessed, focusing on exposure during the mid-1970s. Assessments were made for each of the 214 codes used to describe each individual's occupation during the period 1971–1985/86. After 1985, only 90 job codes were used and a similar matrix was made for the 90 job codes used after 1985.

In the final analysis, categories were combined, resulting in four major exposure groups, as follows: inorganic dust (asbestos, man-made mineral fibres, cement, concrete and quartz); gases and irritants (epoxy resins, isocyanates and organic solvents); fumes (diesel exhaust, metal fume and asphalt); and wood dust. Basic data about the cohort is presented in table 1. As shown in table 2, there is an overlap between the four major exposure groups. Specifically, workers classified as exposed to inorganic dust were also exposed to gases and irritants, and also to fumes.

Statistics

Relative risks (RR) were calculated by the person-year method [26], using the age distribution in the reference group as a control. Person-years were calculated from the calendar year after the first health examination until death, emigration or 31 December 1999, whichever came first. The analyses were stratified according to age in 5-yr groups, smoking habits in four levels and calendar year. Person-years >84 yrs were excluded. Ninety-five per cent confidence intervals (CI) were calculated using a Poisson distribution. The influence of major exposure group, smoking habits and age were analysed using a Poisson regression model [27].

The proportion of COPD among the exposed attributable to work was calculated as attributable proportion (AP) [28]:

$$AP = (RR - 1)/RR \quad (1)$$

The population attributable risk was not calculated, only the attributable risk among the exposed, as this is a cohort not representative of the general population.

Results

Altogether 523 deaths due to COPD occurred among workers exposed to any airborne exposure and there were 200 deaths among the controls. Using the person-year method, stratifying for smoking and age, there was a slightly increased

Table 1. – Basic data on a cohort of 317,629 male Swedish construction workers divided into the different major exposure groups

Exposure	Subjects n	Person-years n	S %	XS %	NS %	US %	Year of birth	Examination
Controls	116894	2203973	35.7	14.8	46.6	6.9	1944.7±15.4	1979.0±6.5
Inorganic dust	154324	2911313	37.8	14.8	41.8	6.5	1943.5±17.1	1978.3±6.4
Gases and irritants	52434	921677	43.0	13.4	39.1	5.5	1946.7±17.5	1979.9±6.8
Fumes	69657	1319629	33.6	15.2	44.1	7.8	1943.3±15.4	1978.5±6.4
Wood dust	21479	381351	46.2	13.5	35.3	5.8	1946.0±18.2	1979.7±7.2
Any airborne exposure	200735	3746488	38.2	14.6	41.5	6.6	1942.8±17.0	1978.6±6.5

Data are presented as mean±SD unless otherwise stated. Smoking habits at start of follow-up divided into the following. S: smokers; XS: exsmokers; NS: never-smokers; and US: unknown smoking habits. Note that an individual can occur in more than one category.

Table 2. – Data on the overlap between different exposure categories

Major exposure group	Inorganic dust	Gases and irritants	Fumes	Wood dust
Inorganic dust	100	74.5	70	6.2
Gases and irritants	25	100	5	25
Fumes	24	5	100	0
Wood dust	0.7	10	0	100

Data are presented as %.

RR of death due to COPD (RR 1.12 (95% CI 1.03–1.22)) among workers with any airborne exposure as compared to controls. In the different major exposure groups, except exposure to wood dust, there was increased mortality due to COPD. Formal statistical significance was observed among those exposed to inorganic dust (RR 1.16 (95% CI 1.05–1.28)) and among those exposed to fumes (RR 1.22 (95% CI 1.04–1.42)) (table 3).

When restricting the analyses to never-smokers, the mortality due to COPD among workers with any airborne exposure further increased (RR 2.11 (95% CI 1.43–3.00)). In addition, the RRs increased and remained significant among those exposed to inorganic dust, gases and irritants, and to fumes (table 4).

The results of the Poisson regression analyses are shown in tables 5 and 6. When analysing the whole cohort, the increased mortality risk remained among those exposed to inorganic dust (hazard ratio 1.10 (95% CI 1.06–1.14)), but the risk declined among the groups exposed to gases and irritants, and to fumes. When restricting the analyses to never-smokers, there was an increased risk among those exposed to inorganic dust, gases and irritants, and to fumes.

The fraction of COPD attributable to work among the exposed was estimated as 10.7% overall and 52.6% among never-smokers.

Table 5. – Hazard ratios (HR) for chronic obstructive pulmonary disease among male construction workers according to different exposures based on a Poisson regression analysis

Predictor (exposure)	HR (95% CI)
Inorganic dust	1.10 (1.06–1.14)
Gases and irritants	1.02 (0.96–1.08)
Fumes	0.98 (0.93–1.03)
Wood dust	0.77 (0.64–0.92)
Age[#]	5.47 (5.30–5.64)
Current smoking	15.3 (11.2–20.8)
Ex-smoking	2.94 (2.09–4.13)
Unknown smoking habits	6.87 (4.95–9.56)

CI: confidence interval. [#]: HR corresponds to an increase in age of 10 yrs.

Table 6. – Hazard ratios (HR) for chronic obstructive pulmonary disease among male construction workers according to different exposures based on a Poisson regression analysis among never-smokers

Predictor (exposure)	HR (95% CI)
Inorganic dust	2.30 (1.07–4.96)
Gases and irritants	3.85 (2.50–5.94)
Fumes	4.44 (2.80–7.04)
Wood dust	1.35 (0.11–17.2)
Age[#]	15.1 (9.4–24.2)

CI: confidence interval. [#]: HR corresponds to an increase in age of 10 yrs.

Discussion

The main finding in this study was an increased mortality from COPD among construction workers exposed to inorganic dusts, as compared to other unexposed construction workers. In this cohort of male construction workers, the proportion of COPD that was attributable to work was considerable, with an attributable fraction of almost 11%. The meaning of this is that reduction of workplace exposures to dust, gases and fumes would prevent one out of ten deaths due to COPD, regardless of other risk factors, such as cigarette smoking.

The main strengths of the present study are that it is a prospective, longitudinal study of a large cohort and that smoking habits have been recorded. In addition, the cohort is based on a register with a high complete coverage of the Swedish construction industry. Finally, an internal control group with known smoking status has been used.

There may be some misclassification of diagnoses between asthma and COPD. According to ICD 8, priority regarding the underlying cause of death was given to COPD if it was

Table 3. – Mortality from chronic obstructive pulmonary disease (COPD) among male construction workers according to exposure to different agents

Exposure	Deaths from COPD n	Relative risk 95% CI	Individuals n	Person-years
Controls	200	1.0	116894	2203973
Inorganic dust	423	1.16 (1.05–1.28)	154324	2911313
Gases and irritants	127	1.18 (0.98–1.41)	52434	921677
Fumes	165	1.22 (1.04–1.42)	69657	1319629
Wood dust	35	0.77 (0.53–1.07)	21479	381351
Any airborne exposure	523	1.12 (1.03–1.22)	200735	3746488

CI: confidence interval. A man can be included in more than one exposed category. Analysed with the person-years method stratified for smoking and age.

Table 4. – Age-adjusted mortality (relative risk) in chronic obstructive pulmonary disease (COPD) among male construction workers according to different exposures among never-smokers

Exposure	Deaths in COPD n	Relative risk 95% CI	Individuals n	Person-years
Controls	7	1.0	49920	861028
Inorganic dust	28	2.38 (1.59–3.44)	57927	1003006
Gases and irritants	11	2.92 (1.46–5.22)	22406	356566
Fumes	10	2.72 (1.31–3.68)	23348	400167
Wood dust	1	0.53 (0.01–2.94)	9840	156078
Any exposure	31	2.11 (1.43–3.00)	76103	1295542

CI: confidence interval. A man can be included in more than one exposed category. Analysed with the person-years method stratified for smoking and age.

mentioned together with asthma, regardless of the cause of death on the death certificate [29]. This priority rule has been excluded in subsequent classifications, meaning that since 1986 the underlying cause of death has been coded according to the death certificate. As a result of this, for the period before 1987, some asthmatics may have been coded as having died from COPD, but this misclassification is probably independent of the subject's occupation.

It is probable that the workers in the control group had been exposed to dust, albeit at lower levels than in the exposed group, which may underestimate the risks. Furthermore, the current authors have not been able to estimate the cumulative exposure index, since the exposure is only based upon occupation reported at the first health examination. It should, however, be noted that, in general, Swedish construction workers remain in the same occupation within the construction industry. In those examined at least twice, >70% had the same occupation after 5 yrs. The job-exposure matrix was based on exposure estimations from the 1970s. Several of the workers (in particular those who were older) had probably worked in the construction industry since the 1930s, 1940s or 1950s. Therefore, exposure to asbestos, for example, may have occurred without being taken into account in the job-exposure matrix. In particular, the current authors believe that previous asbestos exposure among plumbers may be the reason for the increased risk of dying from COPD among workers exposed to metal fume, as the group of workers exposed to metal fume consisted mainly of plumbers (80%). There was also a considerable overlap between those classified as exposed to inorganic dust and those exposed to fumes, and to gases and irritants. In the Poisson regression model it was only exposure to inorganic dust that remained associated with COPD mortality when other major exposure groups were introduced into the model.

The classification of smoking habits in the study was not time dependent. Hence, the results from the whole group can be criticised, as different exposure groups may have different cumulative exposure to tobacco smoke, *i.e.* different pack-years. However, this has been overcome by only analysing never-smokers, which was possible because of the size of the data set. Nevertheless, smoking habits were only reported at the start of follow-up and, therefore, there is a possibility that the fraction of never-smokers who started to smoke was different in the different exposure categories. However, if the analysis among never-smokers was restricted to those with smoking information from repeated health examinations, the relative risk for any airborne exposure did not change (RR 2.11).

Although there is a large body of scientific literature describing studies of COPD concerned with impaired lung function and/or symptoms, this is, to the best of the authors' knowledge, the largest study investigating the relationship between COPD mortality and occupational airborne exposures. The power of the study made it possible to separately analyse different smoking categories. HNIZDO *et al.* [30] recently found an increased risk for COPD among US construction workers (odds ratio (OR) 1.2), especially among never-smokers (OR 3.4). In the Dutch Zutphen study, an increased risk for nonspecific lung disease was found among construction and cement workers [31]. The present results corroborate those from previous studies, indicating an increased risk for COPD among workers exposed to dust, such as asbestos, man-made mineral fibres, mineral dust and concrete dust. However, it should be stressed that the dust exposure is construction related, *i.e.* the results cannot be directly applied to the general population.

The mechanism for the relationship between dust exposure and COPD remains unclear. CHURG and coworkers [32–33], in two separate studies, have previously reported the presence of fibrosis in the small airways among autopsy cases with a previous history of occupational dust exposure. Increased elastic recoil pressure among workers exposed to man-made mineral fibres has also been described [34]. Taken together, these findings indicate that occupational exposure to dust, especially mineral dust, causes a scarring or fibrosis in the small airways.

When analyses were restricted to never-smokers, the increased mortality remained, which to some extent is contrary to other studies [2], although it is in accordance with others [30]. The attributable fraction among never-smokers with any airborne exposure was high, 53%. HNIZDO *et al.* [30] also found a high attributable fraction, 31%, among never-smokers. However, their study was based on the general population and the attributable fraction was a population-based estimate. In South African studies of gold miners, the results indicated a multiplicative interaction between smoking and dust exposure [35, 36]. The data presented in this study rather suggest an additive component in the relationship between smoking and occupational dust exposure. However, it should be noted that none of the nonsmokers in the South African cohort died of COPD. It should be stressed that the estimate for COPD among never-smokers in the current study is based on 31 exposed and deceased subjects.

Two studies from the US general population have recently been published and show a population-attributable fraction of ~20% for occupation and COPD [30, 37]. This is higher than was found in the current study, especially considering only construction workers were included here. The disparity could be due to differences in exposure between the countries, but also due to the fact that other "nonexposed" construction workers were used as controls. In the general population, "unexposed" subjects probably have a lower exposure to dust than the controls used in this study. Furthermore, the US studies may also include other types of occupational exposure not occurring among construction workers.

In conclusion, occupational exposure to dust increases mortality due to chronic obstructive pulmonary disease, even in never-smokers.

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